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Parasitism, developmental plasticity and bilateral asymmetry of wing feathers in alpine swift, *Apus melba*, nestlings

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The hypothesis that developmental instability is a cost of developmental plasticity is explored using the alpine swift (*Apus melba*) as a model organism. In a previous study, experimentally parasitized nestlings showed a reduced wing growth rate in the first half of the rearing period when parasites were abundant (i.e. peak infestation) and an accelerated growth rate (i.e. compensatory growth) in the second half when parasites decreased in number. This suggests that alpine swifts are able to adjust growth rate in relation to variation in parasite loads. Because developmental plasticity may entail fitness costs, the energy required to sustain compensatory growth may be invested at the expense of developmental stability, potentially resulting in larger deviations from symmetry in paired, bilateral traits (i.e. fluctuating asymmetry, FA). This hypothesis predicts higher FA in parasitized than deparasitized nestlings because of compensatory growth, and hence individuals sustaining the highest level of compensatory growth rate should exhibit the highest FA levels. Another non-mutually exclusive hypothesis argues that parasites directly cause FA by diverting energy required by host for maintenance and growth, and predicts that individuals suffering the most from parasitism during peak infestation should exhibit the highest FA levels. The present study shows that wing feathers of experimentally parasitized nestlings were more asymmetrical than those of experimentally deparasitized ones 50 days after hatching. Furthermore, in parasitized individuals FA was negatively correlated with wing growth rate during the period of peak infestation but not during the period of compensatory growth. These findings suggest that developmental homeostasis is more sensitive to parasites than to compensatory growth.

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In species facing ecological factors that vary strongly in time and space, individuals may be selected to display different growth trajectories. Developmental plasticity may be adaptive in allowing individuals to adjust growth rate according to prevailing environmental conditions (Schew and Ricklefs 1998, Metcalfe and Monaghan 2001). For example, under bad rearing conditions offspring may reduce growth rate and accelerate it once the conditions improve (i.e. compensatory growth, Metcalfe and Monaghan 2001) and/or spend more time to complete full development and maturation (i.e. delayed maturation, Metcalfe and Monaghan 2001). Because body size is a major life history trait (Stearns 1992), compensatory growth and delayed maturation can be adaptive in permitting individuals to recover from temporarily stressful rearing conditions and to reach a similar final body size as individuals reared under optimal conditions. The fact that adaptive developmen-

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tal plasticity seems to have evolved in a restricted number of animal species suggests that substantial costs are associated with this process (Arendt 1997, DeWitt et al. 1998, Schew and Ricklefs 1998, Metcalfe and Monaghan 2001). The nature of these costs, the time scale over which they are paid and the underlying mechanism remain poorly understood (Metcalfe and Monaghan 2001). Detailed studies on these issues are required to determine the factors that constrain the evolution and maintenance of developmental plasticity.

Here, we investigated if developmental instability is a cost of compensatory growth. Developmental instability is the result of small and random errors from a predetermined optimal developmental trajectory and phenotype (Waddington 1942, Palmer 1994). If these developmental errors are not neutralised by processes related to developmental homeostasis, they may accumulate, be detectable at the morphological level, and ultimately penalise individuals (reviewed by Møller and Swaddle 1997). Such developmental errors are commonly measured as fluctuating asymmetry (FA), the random deviations from perfect symmetry in bilateral, paired morphological traits (Van Valen 1962). Compensatory growth may increase the level of FA because a large amount of energy is required to sustain fast growth, and hence less energy may be devoted to developmental stability resulting in more developmental mistakes, and hence larger deviations from symmetry in paired, bilateral traits. Therefore, stressful rearing conditions may lead to higher levels of FA for two reasons. First, poor rearing conditions may have a direct effect on FA because less energy may be devoted to the maintenance of a symmetric phenotype when individuals are stressed. This hypothesis predicts a negative relationship between FA and growth rate during the period of stress when individuals display a lower than average growth rate. A role of stressful rearing conditions on FA has been experimentally demonstrated in a wide range of organisms (e.g. insects: Hosken et al. 2000; fishes: Mazzi and Bakker 2001; birds: Swaddle and Witter 1994, Ohlsson and Smith 2001, Brown and Brown 2002; mammals: Sciulli et al. 1979, Folstad et al. 1996), under various environmental stress factors (temperature: Hosken et al. 2000, food: Sciulli et al. 1979, Swaddle and Witter 1994, Ohlsson and Smith 2001, chemical agents: Mazzi and Bakker 2001; parasites: Folstad et al. 1996, Brown and Brown 2002, reviewed by Møller 1996). Second, stressful rearing conditions may have an indirect effect on FA when individuals display a higher than average growth rate after the period of stress has passed (i.e. compensatory growth). This hypothesis predicts a positive relationship between FA and growth rate during the period of compensatory growth. The aim of the present study is to test these two non-mutually exclusive hypotheses.

In a previous study (Bize et al. 2003a), we showed that alpine swift (*Apus melba*) nestlings face blood-sucking louse-flies *Crataerina melbae* (Diptera, Hippoboscidae) mainly during the first half of their development (the first 30 days of life) causing experimentally infested individuals to grow wings at a lower than average rate. In the second half (between 30 and 60 days of age), parasites become less numerous, and previously infested nestlings were shown to accelerate wing growth rate, allowing them to reach a similar final body size as experimentally parasite-free nestlings. As the peak of parasite infestation (i.e. stressful conditions) and compensatory growth do not occur at the same developmental period (first versus second half of the rearing period), it is possible to disentangle the relative contribution of parasites and compensatory growth to FA. For this purpose, we measured growth rate during the period of peak infestation and compensatory growth, and FA in wing feathers of 50 days old (fledging occurs at 50–70 days of age) experimentally parasitized and deparasitized nestlings. We first tested if parasitized nestlings had larger asymmetry in wing feathers than deparasitized ones. Then, to tease apart the relative effect of parasites and compensatory growth to FA in wing feathers, we used multiple linear regressions where growth rates of wing length during the period of peak infestation (between 20 and 30 days of age) and during the period of growth compensation (between 40 and 50 days of age) were entered as factors and FA in wing feathers of 50 days old nestlings as dependent variable. This model allowed us to test two non-mutually exclusive hypotheses. The first hypothesis proposes that compensatory growth causes FA because the energy required for a fast growth is invested at the expense of developmental homeostasis. This hypothesis predicts that, in experimentally parasitized nestlings, growth rate of wing length measured during the period of compensatory growth is positively related to FA. The second hypothesis advocates that parasites cause FA because they divert the energy required by host for body maintenance and growth. This hypothesis predicts that growth rate of wing length measured during peak parasite infestation is negatively related to FA. No similar relationships between FA and wing growth rates are expected in experimentally deparasitized nestlings, since the removal of ectoparasites is supposed to improve rearing conditions and relax constraints.

Material and methods

The alpine swift

The alpine swift is a 90 g migrant colonial apodiform bird that feeds exclusively on insects caught on the wings. It is socially monogamous and reproduces in colonies counting a few (< 5) to several hundred breeding pairs

located in holes of cliffs and tall buildings. One clutch of 1–4 eggs (mean is 2.6) is laid per year, and both parents incubate it for approximately 20 days (Arn 1960). Offspring fledge at an age of 50 to 70 days, and after fledging they do not return to the colony until the following year (Arn 1960). Alpine swifts are heavily parasitized by the 7 mm long blood-sucking louse-fly *C. melbae* that feeds exclusively on this host species (Tella et al. 1998).

Manipulation of louse-fly load

Fieldwork was conducted in 2001 in a colony of 50 breeding pairs located under the roof of a clock tower in Solothurn, Switzerland (47°12′ N, 7°32′ E). From the middle of May onwards, nests were visited every other day to determine the date of clutch completion, and daily around hatching to determine the hatching date of the first egg (day 0). We matched pairs of nests with the criteria that paired nests contained a similar number of 10-days-old nestlings (Wilcoxon rank sum test, $Z = -0.35$, $n = 19$ pairs of nests, $P = 0.72$) that hatched at a similar date ($Z = 0.13$, $n = 19$, $P = 0.89$) and carried a similar number of louse-flies ($Z = -0.59$, $n = 19$, $P = 0.56$). When nestlings were 10 days old, we extracted and counted all ectoparasites from each pair of nests. We started at 10 days because *C. melbae* are rarely found on nestlings before this age (Roulin et al. 2003a). We then, transferred all parasites collected on one of the broods, chosen randomly and denoted 'deparasitized brood', into the other brood called 'parasitized brood'. *Crataerina melbae* is highly mobile, and hence deparasitized nests were naturally re-infested by ectoparasites. We thus repeated the manipulation of ectoparasite loads every five days until nestlings were 50 days old. Though our manipulations consisted of adding parasites to already infested nests, the number of louse-flies per parasitized nestling remained within the natural range of infestation, as observed in other Swiss colonies (Roulin et al. 1998, Bize et al. 2003a). After 20 days of age, alpine swift nestlings start to invade neighbouring families, and are frequently adopted by foster families (Arn 1960, Bize et al. 2003b). To prevent adoption, we checked all nests daily between 20 and 50 days after hatching, and returned each nest-switcher immediately after its discovery to its natal nest. At each transfer of parasites between pairs of nests, nestling wing length and body mass were measured to the nearest millimetre and 0.1 g, respectively.

Measurement of asymmetry

Asymmetry in the wing feathers was measured at 50 days after hatching. For this purpose, two adjacent primary feathers were laid on top of one another and the distance from tip to tip was measured (Roulin et al. 2003b). There are ten primary feathers per wing (P_1 to P_{10}), and thus nine distances between two adjacent feathers to measure ($D_1 = |P_1 - P_2|$; $D_2 = |P_2 - P_3|$; and so on to $D_9 = |P_9 - P_{10}|$). Using a ruler, the same person (P.B.) measured twice these nine distances to the nearest 0.5 mm on the same day. We calculated the absolute asymmetry for each distance as $|Di_{left} - Di_{right}|$, and then summed these nine absolute values. Our method of measuring FA has been demonstrated to provide a reliable estimate of true FA in the length of primary feathers in the barn owl *Tyto alba* (Roulin et al. 2003b). Our FA measurements were restricted to asymmetry in the length of primary feathers, because tips of tail feathers were frequently damaged at 50 days after hatching, and the length of the tarsus was already fixed before we started to manipulate ectoparasite loads, i.e. 10 days after hatching (P.B. pers. obs.).

Statistical analyses

Only nestlings that reached an age of at least 50 days were included in the analyses, which restricted sample size to 19 deparasitized broods and 18 parasitized ones (one parasitized breeding pair failed). We excluded one deparasitized and four parasitized nestlings from the analyses of asymmetry because the tips of some of their feathers were broken, and it was impossible to discriminate FA from unusual feather wear and damage. The relative importance of true asymmetry relative to measurement errors on our FA-measurements was assessed for each of the nine distances Di as proposed by Palmer and Strobeck (1986). The statistical characteristics of signed asymmetry were further assessed by using one sample t-tests to investigate directional asymmetry, and using measures of platykurtosis to investigate departures from normality (Palmer 1994). We corrected significance levels for the number of traits examined using the Bonferroni method (Rice 1989). Absolute FA-estimates were Box-Cox transformed to achieve normality. Growth rates (R) of wing length (W) between 20 and 30 days of age and between 40 and 50 days were calculated with the formulas: $R_{20-30} = (W_{30} - W_{20})/10$ and $R_{40-50} = (W_{50} - W_{40})/10$. For each treatment (parasitized vs deparasitized broods), we used a multiple linear regression analysis to disentangle the relative contribution of growth rate of wing length R_{20-30} (denoted as 'wing growth rate during peak infestation') and R_{40-50} ('wing growth rate during the period of compensatory growth') to FA in wing feathers of 50 days old nestlings. Hatching date and brood size were included as covariates in our multiple linear regression analyses between FA and wing growth rates, since these two factors explained a significant part

of the variance in wing growth rates. Individuals were used as the unit of analysis to examine the statistical properties of our FA measurements. Mean sibling values were used in all other analyses. The JMPIN statistical package was used for all statistical analyses (Sall and Lehman 1996). P-value smaller than 0.05 are considered as significant.

Results

Statistical description of asymmetry variation

Asymmetry in wing feathers could be significantly discerned from measurement errors in all Di, and was fluctuating as indicated by the lack of significant departure from zero of mean signed FA and no obvious departure from normality in all Di, except the difference D_1 (Table 1). Deviation from normality of this trait was attributable to one outlier, because after omitting this nestling, FA followed a normal distribution. We included however this outlier in all further analyses, since rare cases of extreme developmental instability are important in FA studies (Møller and Swaddle 1997). The inclusion of this outlier did not alter our final results. We did not apply size correction of FA, because FA was not significantly correlated to trait size (Pearson correlation between absolute FA and mean D, $n = 72$ nestlings, $r = -0.06$, $P = 0.63$).

Parasitism, wing growth rate and asymmetry

Parasitized nestlings used growth compensation to recover from the effect of parasites on the development of their wings as indicated by the negative correlation between wing growth rates measured during peak infestation (i.e. 20–30 days of age) and 40–50 days of age (Pearson correlation, $r = -0.53$, $n = 18$ broods, $P = 0.02$, Fig. 1A). In deparasitized broods, no similar relationship was found between growth rates of wing length at 20–30 days of age and at 40–50 days of age ($r = -0.23$, $n = 19$, $P = 0.37$, Fig. 1B). Correlation coefficients were statistically similar in parasitized and deparasitized broods ($Z = 0.99$, $P = 0.33$).

Our manipulation of ectoparasite load significantly affected the level of FA in wing feathers measured at day 50 after hatching (Student t-test, $n = 37$, $t = 2.73$, $P = 0.01$), with parasitized nestlings being 1.7 times more asymmetrical than deparasitized ones (Fig. 2). A multiple linear regression showed that, in parasitized broods, FA in wing feathers of 50-days-old nestlings was not related to wing growth rate measured during the period of growth compensation taking place at 40–50 days but negatively with wing growth rate measured during peak infestation (Table 2). In deparasitized broods, FA was neither associated with wing growth rate measured

Table 1. Descriptive statistics for asymmetry in wing feathers of alpine swift nestlings. Mean Di, signed and absolute asymmetry, and percentage of measurement error relative to asymmetry are shown along with results of tests for directionality, measurement errors and normality.

Trait*	n	Distance mean (mm)	Signed FA mean (mm)	Absolute FA mean (mm)	Error† (%)	One-sample t-test‡	F-test§	Skewness g1	Kurtosis g3
D_1 ($P_1 - P_2$)	72	13.2	0.56	0.97	0.5	1.18	192.0¶	8.08¶	67.43¶
D_2 ($P_2 - P_3$)	72	16.3	−0.06	0.57	12.9	−0.56	7.8¶	−0.36	1.74
D_3 ($P_3 - P_4$)	72	18.2	0.06	0.60	16.3	0.57	6.1¶	−0.05	0.51
D_4 ($P_4 - P_5$)	72	20.6	0.22	0.57	15.9	2.33	6.3¶	−0.16	0.90
D_5 ($P_5 - P_6$)	72	22.8	0.31	0.76	11.9	2.85	8.4¶	−0.19	−0.44
D_6 ($P_6 - P_7$)	72	22.3	−0.01	0.89	3.0	−0.07	31.6¶	−5.24	37.62
D_7 ($P_7 - P_8$)	72	16.0	0.36	0.83	4.0	1.69	24.8¶	−6.09	46.43
D_8 ($P_8 - P_9$)	72	5.0	0.07	0.42	19.7	0.74	5.1¶	3.39	20.08
D_9 ($P_9 - P_{10}$)	72	3.6	0.00	0.40	20.4	0.01	4.9¶	−0.17	1.67

*Di is the distance from tip to tip between two proximate primary feathers (Pi − Pi+1).
†Measurement error relative to FA (Palmer and Strobeck 1986).
‡Test if mean signed FA is different from zero.
§Test if FA > measurement error.
¶Significant at $P < 0.05$ after sequential Bonferroni adjustment.

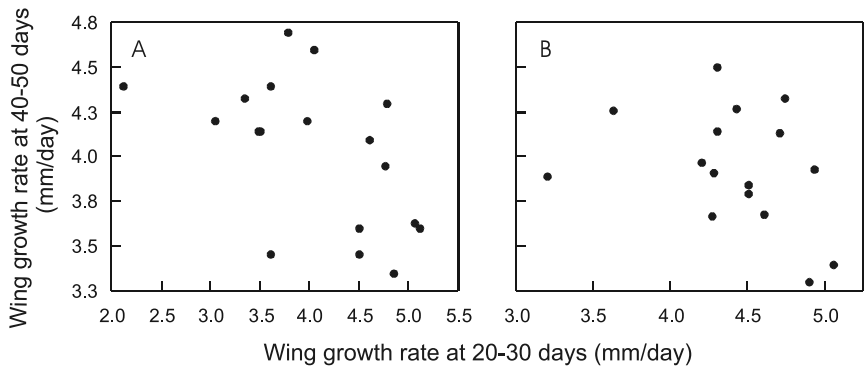

Fig. 1. Relationship between wing growth rate measured during peak parasite infestation (day interval 20–30) and during the period of compensatory growth (day interval 40–50) in (A) parasitized and (B) deparasitized alpine swift broods.

during peak infestation nor during the phase of compensatory growth (Table 2).

Discussion

In a previous study, we have shown that experimentally parasitized alpine swift nestlings grow their wings at a lower than average rate during the first half of their development. In the second half, parasites become less numerous, and parasitized nestlings accelerate wing growth rate to reach a similar final size as deparasitized ones (Bize et al. 2003a). The present study demonstrates that, although wing length at fledging was independent of our ectoparasite treatment, wing feathers of experimentally infested individuals were more asymmetrical.

Two hypotheses can explain why parasitized nestlings grow more asymmetrically than deparasitized ones. First, parasites may have diverted energy from their hosts that would otherwise have been allocated to growth and body maintenance, leading to greater levels of FA in parasitized nestlings. Second, compensatory growth may require a significant amount of energy that could not be invested in developmental homeostasis. The finding that FA in parasitized nestlings was negatively correlated with wing growth rate during peak infestation but not during the period of compensatory growth suggests a direct role of parasitism in processes linked to developmental stability. This is in agreement with previous studies demonstrating that both parasites (Brown and Brown 2002, reviewed by Møller 1996) and the activation of the immune system via an artificial injection of a non-pathogenic antigen (Fair et al. 1999, Whitaker and Fair 2002) increase the level of FA in their hosts. Thus, up-regulation of the immune system may destabilise the development of parasitized individuals, and explain the general association found between parasitism, fitness and FA (Møller 1996, Møller and Swaddle 1997).

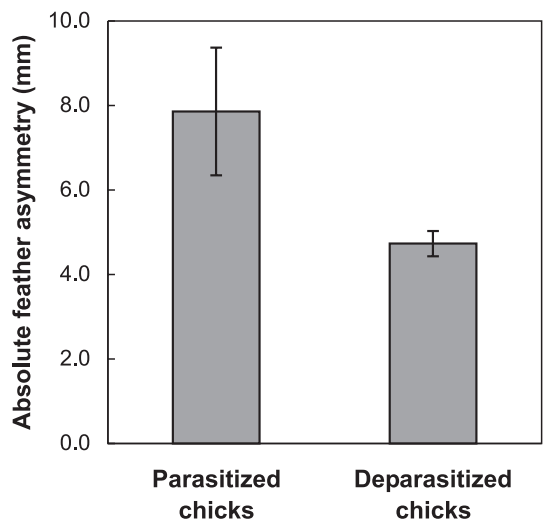

Fig. 2. Mean (±1SE) asymmetry in primary feather length of parasitized and deparasitized alpine swift nestlings.

No relationship between compensatory growth and FA was found, but this last result should be treated with caution for three reasons. First, our investigation of a potential link between compensatory growth and developmental stability is correlative. Thus, it cannot be excluded that parasitized nestlings differed in quality (Reznick et al. 2000), the best ones being able to both grow fast and maintain a symmetric phenotype while the poorest ones grew both slowly and asymmetrically. Differences between individuals in quality may therefore have hidden a trade-off at the individual level between compensatory growth and FA. An experimental demonstration that growth acceleration after a period of low growth rate is traded against developmental stability would require the comparison of FA between two groups of randomly chosen individuals that differ only in the presence or absence of compensatory growth (and not within one group of individuals that all perform compensatory growth). Such experimental design may be very difficult to achieve, because one has to induce compensatory growth without altering environmental conditions. The present study is therefore a first step in

Table 2. Multiple linear regressions between FA and growth rate of wing length in parasitized and deparasitized alpine swift broods. FA was measured at 50 days of age, and wing growth rate at peak infestation (day interval 20–30) and at the period of compensatory growth (day interval 40–50). Interactions were non-significant ($P > 0.51$), and thus were excluded from the final models. F-ratio, R^2 statistic, slope (b) of the linear regression and statistical significance (P) are presented.

Trait	Sample size	Wing growth rate							
		at day interval 20–30				at day interval 40–50			
		F	R^2	b ($\pm$1 SE)	P	F	R^2	b ($\pm$1 SE)	P
FA in parasitized broods	18	6.31	0.25	−1.85 (0.74)	0.03	0.11	0.00	−0.43 (1.30)	0.34
FA in deparasitized broods	19	0.68	0.04	1.41 (1.71)	0.42	0.01	0.00	−0.07 (1.54)	0.92

disentangling the two possible confounding sources of developmental instability

Second, the amount of stress required to disturb developmental homeostasis may vary during the course of development, with early phases being more sensitive to stress factors (Aparicio 2001, Ohlsson and Smith 2001). For instance, pheasant (*Phasianus colchicus*) chicks grew asymmetric tarsi when fed with a low protein diet during their first three weeks of life, whereas similar manipulations performed latter in their development had no effect on symmetry in tarsus length (Ohlsson and Smith 2001). Since compensatory growth usually occurs late in development, the relative effect of compensatory growth to FA may be less detectable than the effect of stress factors occurring at an earlier stage.

Finally, since we assessed FA only in wing feathers, it could not be excluded that fast growth of wing length was traded against developmental homeostasis of other traits. For instance, within-broods nestling barn swallows (*Hirundo rustica*) with the largest rate of feather growth had the lowest rate of tarsus and body mass growth (Saino et al. 1998). Thus, further studies should preferably assess the relationship between FA and compensatory growth in a wide range of morphological traits.

In conclusion, the present study demonstrates that experimentally parasitized nestlings grew more asymmetrical wing feathers than experimentally deparasitized ones. We found no support for a link between compensatory growth and developmental stability, and hence high level of FA in parasitized individuals occurred because parasites diverted energy that would be otherwise allocated to developmental stability. Although in rats selection lines for fast and slow intrinsic growth have demonstrated that developmental errors can increase with fast growth (Leamy and Atchley 1985, Shakarad et al. 2001), it remains unclear whether episodes of fast growth during a limited period of time can also increase FA (Møller 1999). Further studies are required to determine what are the costs incurred by compensatory growth and developmental plasticity.

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